Yong-jie Zhang

List of Publications by Year
in descending order

32 papers	5,317 citations	201674 27 h-index	414414 32 g-index
35 all docs	35 docs citations	35 times ranked	4834 citing authors

#	Article	IF	Citations
1	Aberrant cleavage of TDP-43 enhances aggregation and cellular toxicity. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 7607-7612.	7.1	523
2	Antisense transcripts of the expanded C9ORF72 hexanucleotide repeat form nuclear RNA foci and undergo repeat-associated non-ATG translation in c9FTD/ALS. Acta Neuropathologica, 2013, 126, 829-844.	7.7	506
3	TDP-43 pathology disrupts nuclear pore complexes and nucleocytoplasmic transport in ALS/FTD. Nature Neuroscience, 2018, 21, 228-239.	14.8	404
4	Progranulin Mediates Caspase-Dependent Cleavage of TAR DNA Binding Protein-43. Journal of Neuroscience, 2007, 27, 10530-10534.	3.6	339
5	C9ORF72 repeat expansions in mice cause TDP-43 pathology, neuronal loss, and behavioral deficits. Science, 2015, 348, 1151-1154.	12.6	332
6	Discovery of a Biomarker and Lead Small Molecules to Target r(GGGGCC)-Associated Defects in c9FTD/ALS. Neuron, 2014, 83, 1043-1050.	8.1	289
7	Aggregation-prone c9FTD/ALS poly(GA) RAN-translated proteins cause neurotoxicity by inducing ER stress. Acta Neuropathologica, 2014, 128, 505-524.	7.7	284
8	C9ORF72 poly(GA) aggregates sequester and impair HR23 and nucleocytoplasmic transport proteins. Nature Neuroscience, 2016, 19, 668-677.	14.8	268
9	Poly(GR) impairs protein translation and stress granule dynamics in C9orf72-associated frontotemporal dementia and amyotrophic lateral sclerosis. Nature Medicine, 2018, 24, 1136-1142.	30.7	241
10	Human C9ORF72 Hexanucleotide Expansion Reproduces RNA Foci and Dipeptide Repeat Proteins but Not Neurodegeneration in BAC Transgenic Mice. Neuron, 2015, 88, 902-909.	8.1	219
11	Heterochromatin anomalies and double-stranded RNA accumulation underlie C9orf72 poly(PR) toxicity. Science, 2019, 363, .	12.6	181
12	Poly(GP) proteins are a useful pharmacodynamic marker for C9ORF72 -associated amyotrophic lateral sclerosis. Science Translational Medicine, 2017, 9, .	12.4	179
13	Reduced C9ORF72 function exacerbates gain of toxicity from ALS/FTD-causing repeat expansion in C9orf72. Nature Neuroscience, 2020, 23, 615-624.	14.8	157
14	The dual functions of the extreme N-terminus of TDP-43 in regulating its biological activity and inclusion formation. Human Molecular Genetics, 2013, 22, 3112-3122.	2.9	156
15	Mechanisms of toxicity in C9FTLD/ALS. Acta Neuropathologica, 2014, 127, 359-376.	7.7	134
16	Spt4 selectively regulates the expression of C9orf72 sense and antisense mutant transcripts. Science, 2016, 353, 708-712.	12.6	116
17	C9orf72 poly(GR) aggregation induces TDP-43 proteinopathy. Science Translational Medicine, 2020, 12, .	12.4	115
18	Aberrant deposition of stress granule-resident proteins linked to C9orf72-associated TDP-43 proteinopathy. Molecular Neurodegeneration, 2019, 14, 9.	10.8	111

#	Article	IF	Citations
19	p53 is a central regulator driving neurodegeneration caused by C9orf72 poly(PR). Cell, 2021, 184, 689-708.e20.	28.9	104
20	Phosphorylation regulates proteasomal-mediated degradation and solubility of TAR DNA binding protein-43 C-terminal fragments. Molecular Neurodegeneration, 2010, 5, 33.	10.8	103
21	Repetitive element transcripts are elevated in the brain of C9orf72 ALS/FTLD patients. Human Molecular Genetics, 2017, 26, 3421-3431.	2.9	101
22	Misregulation of human sortilin splicing leads to the generation of a nonfunctional progranulin receptor. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 21510-21515.	7.1	82
23	The Hairpin Form of r(G4C2)exp in c9ALS/FTD Is Repeat-Associated Non-ATG Translated and a Target for Bioactive Small Molecules. Cell Chemical Biology, 2019, 26, 179-190.e12.	5.2	80
24	Poly-GR dipeptide repeat polymers correlate with neurodegeneration and Clinicopathological subtypes in C9ORF72-related brain disease. Acta Neuropathologica Communications, 2018, 6, 63.	5.2	79
25	The extreme N-terminus of TDP-43 mediates the cytoplasmic aggregation of TDP-43 and associated toxicity in vivo. Brain Research, 2016, 1647, 57-64.	2.2	44
26	Ribonuclease recruitment using a small molecule reduced c9ALS/FTD r(G ₄ C ₂) Tj ETQq0 0 0 rgBT /Overlock 1	12.4	39
27	Hexanucleotide Repeat Expansions in c9FTD/ALS and SCA36 Confer Selective Patterns of Neurodegeneration InÂVivo. Cell Reports, 2020, 31, 107616.	6.4	37
28	Microglial lysosome dysfunction contributes to white matter pathology and TDP-43 proteinopathy in GRN-associated FTD. Cell Reports, 2021, 36, 109581.	6.4	33
29	The AD tau core spontaneously self-assembles and recruits full-length tau to filaments. Cell Reports, 2021, 34, 108843.	6.4	30
30	Structural Features of Small Molecules Targeting the RNA Repeat Expansion That Causes Genetically Defined ALS/FTD. ACS Chemical Biology, 2020, 15, 3112-3123.	3.4	12
31	C-terminal and full length TDP-43 specie differ according to FTLD-TDP lesion type but not genetic mutation. Acta Neuropathologica Communications, 2019, 7, 100.	5.2	11
32	A Small Molecule Exploits Hidden Structural Features within the RNA Repeat Expansion That Causes c9ALS/FTD and Rescues Pathological Hallmarks. ACS Chemical Neuroscience, 2021, 12, 4076-4089.	3.5	8